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AN EVALUATION OF THE LOWER COVERAGE ANTI-G SUIT WITHOUT AN ABDOMINAL BLADDER AFTER 3 DAYS OF 7° HEAD DOWN TILT

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13. ABSTRACT *(Maximum 200 words)*

During space shuttle reentry, volume-depleted astronauts experience slow-onset, long-duration, low-level +G. In Phase 2, we determined that an extended coverave anti-G suit without an abdominal bladder (Reentry Anti-G Suit (REAGS)) was the most effective anti-G suit in subjects dehydrated with furosemide (Lasix). The present study (Phase 3) verified that REAGS provided effective protection for subjects dehydrated with 7 degrees head-down tilt (HDT). Twelve healthy male subjects were placed at 7 degrees HDT for 3 days, and then subjected to an acceleration profile simulating shuttle reentry while wearing the REAGS suit. Six subjects and their anti-G suit inflated when their eye-level blood pressure (ELBP) fell below 60 mmHg, while six subjects had their suit inflated when they experienced peripheral light dim (PLD). Average REAGS pressure required to maintain ELBp at or above 60 mmHg after HDT was 0.85 psig vs. 1.0 psig in subjects dehydrated with Lasix. One subject in the PLD group experienced (G-LOC); however, this may have been related to the greater cardiovascular stress induced by HDT.

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AN EVALUATION OF THE LOWER COVERAGE ANTI-G SUIT WITHOUT AN ABDOMINAL BLADDER AFTER 3 DAYS OF 7° HEAD DOWN TILT

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Abstract

An earlier phase of this study demonstrated that a full lower torso coverage re-entry anti-G suit without abdominal bladder (REAGS) provided effective protection against slow onset, long duration, low level +Gz acceleration in subjects dehydrated with Lasix. However, the dehydration model used in the early phases of this study may not provide an optimal simulation of the cardiovascular deconditioning associated with space flight. Bedrest studies with head down tilt (HDT) are traditionally considered the best approximation of space flight because this method will reset baroreceptors and cause muscular deconditioning. Therefore, this final phase of our study consisted of subjects initially deconditioned by 3 days of 7° HDT bed rest followed by donning the REAGS and being centrifuged using a simulated Shuttle re-entry profile.

Twelve healthy male subjects, ages 23-41, participated in this study. Seven of these subjects had also participated in earlier phases of this study. For 6 of the 12 subjects, anti-G suit pressure was increased in 0.5 psig increments if eye level blood pressure (ELBP) dropped below 70 mmHg. The second half of the subjects had their G-suits inflated in 0.5 psig increments if they reported 50% peripheral light dimming (PLD). The maximum allowable pressure for either exposure was 2.5 psig, which is the operational limit of the Shuttle anti-G suit controller. Physiological results from these studies are reported.

Methods

Subjects reported to the Armstrong Laboratory Bedrest Facility at 0800. Baseline hematocrits and weights were taken. Subjects were then placed at 7° head down tilt (HDT) for 72 hours. Strict fluid intake and urine output measurements were kept, and subjects were allowed free access to food and drink. Aspirin, Tylenol, and antacids were permitted on an as needed basis. Benadryl (diphenhydramine) could be taken for the initial 48 hours of the study, but not within 24 hours of the centrifuge ride.

At the completion of the 72 hours bed rest, subjects were fitted with the re-entry anti-G suit (REAGS) and transported from the bedrest facility to the centrifuge building while maintaining 7° HDT. They were transferred to a specially designed chair at 7° HDT. The chair was then rotated (accelerated) to an upright position with a 5° tilt back seat angle (the seat angle found on the Shuttle) over a 12-minute period (onset rate of 0.0014 G/s). Once the seat was fully rotated, it was secured in place. The centrifuge was then accelerated to 2.4 +Gz over 8 minutes (onset rate of .003 G/s). A plateau at 2.4 +Gz was maintained for the remaining 15 minutes. At the completion of the plateau, there was a gradual onset run (GOR) of 0.1 G/sec until the subjects lost 100% of their peripheral lights (PLL) or reached 5 +Gz. They were instructed to perform an Anti-G Straining Maneuver (AGSM) during the GOR if PLL occurred to determine the effectiveness of a strain in regaining peripheral vision. After the treatment run, the subjects jogged approximately 0.25 miles on a treadmill to simulate egress.

During the centrifuge exposure, eye level blood pressure (ELBP) was measured using a noninvasive pneumatic finger cuff (Finapres™). The technique for the use of the Finapres during acceleration is described by McKenzie.[4] The finger cuff was placed at heart level, and the measurement was corrected to an eye level reading by use of a water column. For 6 of the 12 subjects, anti-G suit pressure was increased in 0.5 psig increments if ELBP dropped below 70 mmHg. The second half of the subjects had their G-suits inflated in 0.5 psig increments if they reported peripheral light dim (PLD). The maximal allowable pressure for either exposure was 2.5 psig which is the operational limit of the Shuttle controller. Pressure increases were controlled by a computer-driven G-valve. Suit pressure was not increased during the GOR portion of the ride.

All subjects were monitored with a 2-lead ECG and cardiotachometer. Light loss was determined by the use of a light bar positioned 76.2 cm from the subject's face, with the peripheral lights subtending a visual angle of 50°.

Due to differences in experimental design, only limited statistical comparisons were made using the seven subjects who participated in both Phase 2 and Phase 3 of this study. Student's paired t-tests were used to compare the effects of Lasix (Phase 2) with the effects of bed rest (Phase 3). Total body water loss (BWL) and hematocrit changes from baseline were examined.

Results

Total body water loss was estimated as the difference between total fluid intake (both oral and intravenous) and total urine output. The average BWL in the first seven subjects for the Lasix condition was 1546 cc vs. 1480 cc for the bedrest condition (Non significant by paired t-test at the 0.05 level). Individual results are shown in Table 1.

TABLE 1: CORRECTED BODY WATER LOSS

(Statistical analysis was performed on only the first 7 subjects.)

SUBJECT	LASIX	BEDREST
1	715	1590 cc
2	1850	1260 cc
3	1400	2170 cc
4	2175	715 cc
5	1130	2076 cc
6	1405	1840 cc
7	2150	700 cc
average	1546 cc	1479 cc
8		2622 cc
9		530 cc
10		172 cc
11		800 cc
12		1830 cc

Hematocrit (hct) readings were drawn at 4 different times during each exposure. In the Lasix portion, hct was measured at the following time points:
Sample 1L: prior to Lasix administration,
Sample 2L: immediately before the centrifuge ride,
Sample 3L: immediately after exiting the gondola,
Sample 4L: after lying quietly for 20 minutes following the termination of the centrifuge ride.

During the bedrest portion, hematocrits were taken at comparable points:
Sample 1B: before being put at 7° HDT,
Sample 2B: immediately before starting the centrifuge ride while at 7° HDT,
Sample 3B: immediately after completing the centrifuge ride and the treadmill run,
Sample 4B: after lying quietly for 20 minutes following completion of the treadmill run.

Results are shown in Table 2.

TABLE 2: AVERAGE HEMATOCRITS FOR LASIX AND BEDREST

(Sample size varied for each sample due to technical difficulties. The n for each sample is listed in parentheses.)

SAMPLE	LASIX	BEDREST
1	43 (6)	45 (6)
2	45 (7)	50 (7)
3	47 (5)	53 (7)
4	46 (7)	49 (7)

To adjust for different starting points (baselines), the changes from baseline hematocrit were computed at each time interval for statistical analysis. The average changes from the baseline hematocrit are shown graphically in Figure 1. ANOVA statistics indicate a significant difference between sample 2L-1L and sample 2B-1B (p=.0046). However, there is no difference between sample 3L-1L and 3B-1B or 4L-1L and 4B-1B (p=0.1004 and p=0.2075 respectively).

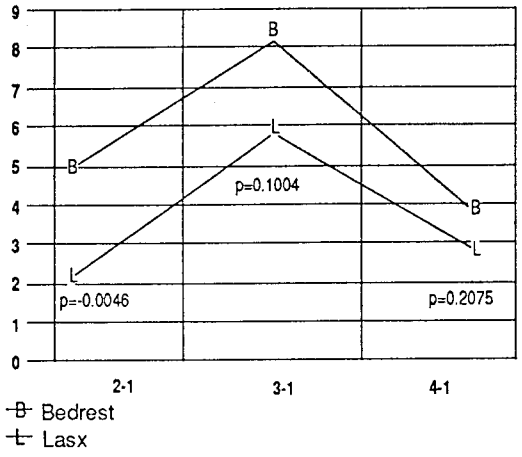

Figure 1: Average hematocrit changes from baseline, all subjects.

Six of the riders had their anti-G suits inflated in 0.5 psig increments if their blood pressure fell below 70 mmHg, while the remaining six had their anti-G suits inflated if they reported PLD. The final pressure required in each condition is listed in Table 3. One subject in the PLD group experienced G-induced loss of consciousness (GLOC) 18 minutes into the centrifuge exposure.

TABLE 3: SUIT PRESSURE IN PSIG REQUIRED IN PHASE 3

SUBJECT	70 mmHg ELBP	PLD
1	2.0 psig	
2	1.0 psig	
3	0.75 psig	
4	0 psig	
5		1.0 psig
6		0 psig
7		GLOC
8	0.5 psig	
9	did not finish ride	
10		0 psig
11		0 psig
12		0.5 psig
average	0.85 psig	0.3 psig

Discussion

The major portion of fluid shifts occurring during space flight are completed within the first few hours of reaching orbit.[5,6,7] These shifts redistribute body fluid and induce free water loss. The resulting dehydration contributes to orthostatic intolerance when the astronauts return to normal earth gravity.[2] Increases in G level encountered during Shuttle re-entry amplify this problem. The degree of impairment is quite variable among crewmembers.

Previous anti-G suit research has centered around the effects of high level, short duration, rapid onset, +Gz acceleration seen in the fighter environment.[1] Unlike fighter aircraft, the maximum +Gz acceleration encountered during Space Shuttle re-entry is less than 1.9. The ability of an anti-G suit to protect against long duration, slow onset acceleration in consonance with dehydration/deconditioning has not been elucidated.

The first two phases of this study determined the effectiveness of anti-G suits of various configurations to protect a subject dehydrated with Lasix. In Phase 2, the suit found to be the most comfortable while still providing acceleration protection was the full lower torso coverage anti-G suit without an abdominal bladder (REAGS). Phase 3 was designed to validate the effectiveness of REAGS in subjects who had undergone 3 days of 7° HDT. Due to the experimental design, statistical evaluations were limited to comparisons between the use of Lasix vs. HDT to model space deconditioning.

The major objective of this phase was to determine if REAGS could maintain ELBP after 72 hours of HDT. The average pressure required to maintain ELBP at 70 mmHg after bedrest was 0.85 psig vs. 1.0 psig after Lasix (NS). The only GLOC incident in this study occurred after 72 hours of HDT. Subject 7, whose suit inflation schedule was based on the symptoms of PLD, experienced GLOC at +2 Gz (18 minutes into the ride). Therefore, the effectiveness of REAGS to prevent GLOC when the suit is inflated on a symptomatic basis is unclear. The failure of this suit may have been, in part, related to its slow inflation rate which simulated the inflation rate on board the Space Shuttle. Thirty to forty-five seconds were required to inflate the suit from 0 to 0.5 psig, and this may not have been rapid enough to prevent a continued fall in blood pressure. A second factor in the GLOC incident may have been related to the unique fluid redistribution in response to bed rest.

Fluctuations of total body water reflect changes in the hydration state, while hct provides an estimate of the percent of total body water retained in the intravascular space. These two parameters provide an approximation of the distribution of body fluids. BWL measurements from Lasix vs. bedrest showed no significant difference between these two methods of dehydration (1546 cc after Lasix vs. 1479 cc after bed rest based on 7 paired subjects). However, the changes in the hct measurements indicated that, although intravascular volume was depleted in both cases, significantly more fluid was lost from the vascular space after bed rest relative to Lasix administration ($p=0.0046$). Therefore, at the initiation of acceleration, the bedrest subjects had less vascular reserve with which to maintain ELBP. Additional fluid was lost from the vascular space following centrifuge ride in both conditions, as evidenced by the further increases in hct in sample 3. While the hct in the Lasix condition did not increase enough to match the hct in the bedrest condition, the difference between the change from baseline seen in the Lasix condition vs. the bedrest condition after the centrifuge ride (3-1) was no longer significant ($p=0.1004$). Following the centrifuge ride, the hcts decrease in both conditions and approach baseline and final samples (4-1) were not significantly different ($p=0.2075$).

These data suggest that bed rest induces greater changes in vascular volume than Lasix but does not induce greater BWL. The vascular redistribution appears to be a direct result of the HDT, as the intravascular and extravascular measurements 20 minutes after returning to +1Gz in the HDT subjects approach those seen in the Lasix subjects. This may explain the GLOC seen after bed rest. Subject 7 had a greater fluid deficit after the Lasix (2150 cc vs 700 cc); however, his hematocrit levels rose to a much higher level after bed rest (Figure 2). After Lasix, the subject had a 9.8% increase in his hematocrit, but following bedrest, his hematocrit went from 42% to 52% (a 24% increase). Sample 3B was actually lower than the sample 2B hct, indicating that the intravascular volume had increased during the centrifuge ride. This suggests that in this subject, bed rest provided a much greater cardiovascular challenge than Lasix, despite a smaller decrease in total body water.

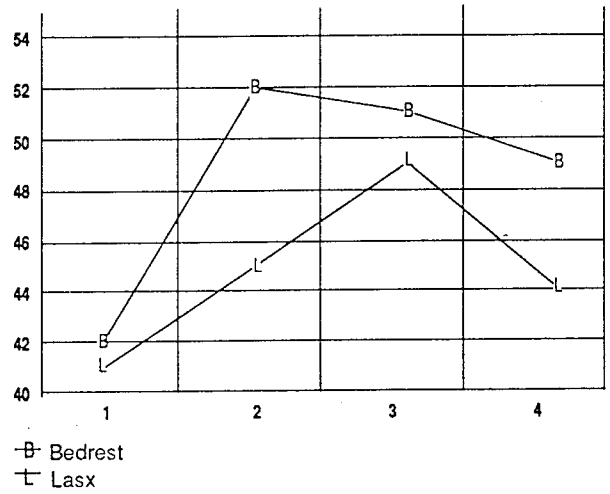

Figure 2: Hematocrit changes for subject 7.

In conclusion, the REAGS suit was able to maintain ELBP in 10 of 11 subjects (data on one subject were not used; his ride was terminated due to motion sickness). The only GLOC occurred in a subject taken to PLD. This GLOC may have been related to the unique pattern of vascular redistribution seen during bed rest and may indicate that bed rest induces a more provocative cardiovascular stress than Lasix administration. This final phase of the study reinforces our prior conclusion in Phase 2: recommended prophylactic inflation of the anti-G suit.

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